

## Case Report: Possible cross-over of Rabbit Haemorrhagic Virus (v2) to a young human boy with subsequent near fatal thrombocytopenia and sepsis \*

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### CASE REPORT

Patient B, a boy aged six years, resident in Germany for just over six months, presented with several days history of mild malaise with muscle aches and a purpuric rash for the previous twenty-four hours. The only other member of the family suffering from malaise and muscle ache at that time was B's mother who remarked that the limb pains were very similar to Dengue fever that both she and the patient had caught in Brazil some two years earlier. B's sister and father were not complaining of any similar condition.

On examination the purplish-red rash was present on Patient B's hands, buttocks, legs and soles of his feet (Figures 1-3). On his feet the rash was confluent covering much of the anterior part of the feet. On examination of his mouth (figure 4) he had petechial spots on the posterior part of his palate. There was slight reddening around his mouth and his nares were reddened.

The "tumbler test" or "glass test" showed that the rash on his body and limbs did not fade with pressure <sup>(1)</sup>. Patient B's parents took him to hospital in view of the high possibility of septicaemia. By the time he reached hospital he was bleeding from his nose, which continued for two days, and he was very much more moribund.

Patient B was immediately admitted to the children's ward and put in isolation with his parent. Blood was taken for blood count, urea and electrolytes and blood culture. The immediate results showed dehydration and profound thrombocytopenia with the platelet count at one thousand or less (1000 being the lowest level measurable). Viral infection was considered to be highly likely as a cause of the problem.

Intravenous cannulation was performed and the patient showed some immediate general improvement with the rehydration.



Figure 1 Petechial rash on Patient B's lower back



Figure 2 Purpuric rash and some bruising on the patient's legs



Figure 3  
Reddening of the anterior  
sole of the foot, pale mid  
sole.



Figure 4  
Reddening around the mouth and within the nares,  
petechial spots on the palate.

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On day 2 the improvement was not sustained and by the end of day 2 patient B was feeling very much weaker in himself.

A diagnosis of idiopathic thrombocytopenia, probably viral in origin, was being considered but on day 3, at forty-eight hours, a gram -ve bacillus was reported to have been cultured from the blood and the patient was put on intravenous antibiotics. This caused a marginal improvement but the antibiotics were changed on day 4 when the full report with sensitivities was obtained. Improvement was then rapid.

The organism cultured was *Acinetobacter lwoffii*, a gram negative bacillus which is found as a normal skin commensal in 40% of people. *Acinetobacter lwoffii* is not usually pathogenic and is normally only implicated in patients with immune suppression or severe malaise. The possibility that it could be a secondary infection following a viral disease affecting both patient B and his mother was being considered when a report was received that one of his two pet rabbits had died. This was followed by the death of the second rabbit within eight hours of the first. On superficial examination after death both rabbits had red eyes due to haemorrhage but no other external signs of abnormality.

The rabbits had been given to Patient B on his birthday two weeks before presentation with the thrombocytopenia. The rabbits were six weeks old at purchase and had shown normal activity until the last few days when they became very docile.

The patient's grandmother, who had been staying with the family, then complained of malaise, muscle ache and some bleeding from her gums. This improved without treatment over a period of a week. Patient B received ten day of IV antibiotics in hospital, followed by oral antibiotics as an outpatient. After taking the full course of oral antibiotics patient B developed further signs including fever and fatigue and required a third course of antibiotics for another ten days. He has now made a full recovery.

## DISCUSSION

According to online reports the most likely cause of sudden demise of two rabbits with no external features of morbidity is Rabbit Haemorrhagic Virus (v2). This

new variant of the Lagovirus, RHDV, was discovered in 2010<sup>(2)</sup>. It has been shown to be a distinctly different virus to the original RHDV. Rabbit Haemorrhagic Virus (v2) is a calicivirus that causes the rabbit to die from internal haemorrhage due to profound thrombocytopenia and the new variant (v2) is spreading rapidly in Europe. It is typical for rabbits to be resistant to the RHDV (v2) for the first eight weeks of life and then succumb.

Recent phylogenetic analysis showed that caliciviruses exhibit high levels of host switching<sup>(3,4)</sup> and reports that RHDV(v2) is host specific to rabbits may prove to be incorrect. RHDV RNA was recently isolated from sympatric wild small mammals, which suggested that the species range of RHDV may not be as limited as previously believed. It is postulated, but not proven, that patient B, his mother and subsequently his grandmother all contracted Rabbit Haemorrhagic Virus (v2) and that patient B was super-infected with the skin commensal which was markedly worsening his prognosis until he received antibiotics.

*Acinetobacter lwoffii*<sup>(5)</sup> is an aerobic gram-negative bacillus occurring as normal flora on the skin and in the oropharynx of between 25 and 40% of the population. It has been identified as the causative organism in urinary tract infections, skin and wound infections, septicaemia, meningitis and gastro-enteritis. However it is usually only pathogenic in immune compromised patients.

Interestingly Patient B's sister, father and grandfather were not affected whilst Patient B, his mother and grandmother all were. All six had been in contact with the rabbits equally. The three who exhibited symptoms had previously caught Dengue fever in Brazil whilst those who did not develop problems had not previously experienced Dengue fever.

It is known that one infection of Dengue fever predisposes to worse signs and symptoms in later Dengue infections, often leading to hemorrhagic problems. One possibility is that Dengue fever also predisposes to infection with Rabbit Haemorrhagic Virus (v2) and in Patient B's case the combined result was immune suppression leading to superinfection with the skin commensal. Alternatively the specific genotype that predisposes

the patient to be affected by Dengue may also predispose to infection by Rabbit Haemorrhagic Virus (v2)<sup>(6)</sup>.

Worryingly in Australia the farmers are purposely spreading Rabbit Haemorrhagic Virus (v2) in an attempt to reduce the invasive rabbit population thus mimicking the way that they spread myxomatosis in previous decades. In Britain the wild rabbit population has fallen by 48% in less than twenty years coinciding with the spread of the viruses and vets have issued a warning that pet rabbits should be vaccinated<sup>(7)</sup>.

## CONCLUSION

A case is reported of haemorrhagic rash from severe thrombocytopenia due to *Acinetobacter lwoffii* septicaemia in a six year-old boy (Patient B). His pet rabbits both died simultaneously, almost certainly from Rabbit Haemorrhagic Virus (v2). It is postulated that infection of Patient B by the Rabbit Haemorrhagic Virus (v2) contributed to the haemorrhagic condition and caused sufficient immunosuppression for the bacterial infection to occur.

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